

A Geometric Model of Mortality and Crop Protection for Insects Feeding on Discrete Toxicant Deposits

TIMOTHY EBERT¹ AND RICHARD DERKSEN²

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ABSTRACT Current theory governing the biological effectiveness of toxicants stresses the dose-response relationship and focuses on uniform toxicant distributions in the insect's environment. However, toxicants are seldom uniformly dispersed under field conditions. Toxicant distribution affects bioavailability, but the mechanics of such interactions is not well documented. We present a geometric model of the interactions between insects and heterogeneously distributed toxicants. From the model, we conclude the following: 1) There is an optimal droplet size, and droplets both smaller and larger than this optimum will decrease efficacy. 2) There is an ideal droplet distribution. Droplets should be spaced based on two criteria: calculate the allowable damage, double this quantity, and one lethal deposit should be placed in this area; and define the quantity of leaf the larva could eat before the toxicant decays below the lethal level and place one lethal deposit within this area. 3) Distributions of toxicant where deposits are sublethal will often be ineffective, but the application is wasteful if deposits contain more than a lethal dose. 4) Insect behavior both as individuals and collectively influences the level of crop production provided by an application. This conclusion has implications for both crop protection and natural plant-insect interactions. The effective utilization of new more environmentally sensitive toxicants may depend on how well we understand how heterogeneous toxicant distributions interact with insect behavior to determine the biological outcome.

KEY WORDS pesticide efficacy, deposit structure, droplet size, application volume

"THE NATURE OF BEHAVIORAL responses reflects toxin apparency, mode of action, and the extent to which sublethal doses influence behavior. The interaction between these behavioral responses to heterogeneously distributed toxins and physiological mechanisms of tolerance has influenced the evolution of insecticide resistance in managed systems and the evolution of plant defensive strategies in natural systems (Hoy et al. 1998)." This statement introduces several key concepts dealing with the way organisms interact with toxicants under field conditions. We develop a simple mathematical model that demonstrates these interactions for a chewing insect feeding on toxicant-treated leaves in an agricultural context. This model focuses on the consequences of changing the dose and distribution of toxicant by changing the numbers and size of deposits and the toxicant per deposit for a chewing insect that only acquires toxicant through feeding; many of the biorational insecticides would fit this model. Although the effects of distribution are well researched in agriculture, the overall

dose transfer process is still poorly understood (Lawrie et al. 1997).

An examination of the literature supports the importance of toxicant distribution in determining toxicant efficacy. Foremost among these are articles showing the importance of droplet size (Munthali 1984; Adams et al. 1987, 1990; Alm et al. 1987; Bryant and Yendol 1988; Maczuga and Mierzejewski 1995; Ebert et al. 1999), with the assumption that droplet size will translate into deposit size: droplets are liquid drops produced by the atomization of the liquid, whereas deposits are the result of these droplets being retained by the target surface. Further evidence of the importance of toxicant distribution comes from the influence of application volume on efficacy (Sopp and Palmer 1990, Falchieri and Cesari 1993, Ebert et al. 1999, Chapple et al. 2000), where application volume is inversely related to toxicant concentration. Although there are many confounding factors obscuring the effects of the individual components in deposit structure, the considerable work on ultralow volume application is a continuing demonstration of the effects of application volume on efficacy (Sopp et al. 1990, Sandhu et al. 1997, Parnell et al. 1999). Furthermore, any study that shows a biological effect from a change in application equipment is, in part, a demonstration of the role of toxicant distribution. This is because a change in application equipment has only two ways that it can influence efficacy: alter retention

¹ Laboratory for Pest Control Application Technology, Department of Entomology, Ohio Agricultural Research and Development Center, The Ohio State University, 1680 Madison Avenue, Wooster, OH 44691.

² Food Agricultural and Biological Engineering, USDA-ARS, 206 Agricultural Engineering Building, Ohio Agricultural Research and Development Center, 1680 Madison Avenue, Wooster, OH 44691.

(thereby changing dose) or alter toxicant distribution. Put simply, "a high-level deposit badly distributed is less efficient than a low-level deposit well distributed" (Frick 1970).

Insect behavior has been implicated as a means whereby insects adapt to new plant defenses and pesticides (Gould 1984; Hoy et al. 1991; Head et al. 1995a,b). A change in behavior correlated with resistance has also been shown for mites (Kolmes et al. 1994). It has been suggested that both behavior and physiology play complimentary roles in the development of resistance to insecticides (Lockwood et al. 1984; Sparks et al. 1989), and by extension this would apply equally to the development of host plant resistance and development of resistance to transgenic crops. Although the role of behavior in resistance has been recognized for some time, we present a simple model that demonstrates how differences in behavior can change toxicant acquisition rates in any system where organisms interact with heterogeneously distributed toxicants. Given that at least some behavior is correlated with genetic traits, this model provides a simple mechanism for selection pressure for certain behaviors.

The model is a strategic model. It is designed to help visualize how a specific part of the dose-transfer process functions for chewing herbivorous larvae acquiring toxicant through feeding by focusing on individual outcomes: one larva on one leaf. It is not a tactical model designed to make numerical predictions about the outcome of a specific plant-pest-toxicant interaction. The benefit of this model is that it provides a rationale for odd reports from the field and shows why these results could be real as opposed to an artifact of poor science or random biological variability, for example, efficacy and dose are not related (Holly 1952, Robinson and Garnet 1984), even though large numbers of laboratory tests show a strong relationship between dose and insect mortality.

The model explores the role of toxicant distribution in determining efficacy. It explains why uniform distribution of toxicant may not be desirable even if it becomes achievable. It explains why many laboratory studies show the efficacy benefit of small droplets, but field studies may not. Gaining a better understanding of how the dose transfer process works may improve both laboratory and field experimental designs and foster innovative approaches to pest management.

Methods and Model. We start with some initial assumptions that we can then remove as the model develops: 1) We model insect larvae chewing on a leaf surface. 2) The insect starts feeding at a random location on the leaf. If it moves, it does so randomly and resumes feeding at a new location chosen randomly. 3) The toxicant only works through ingestion, and the only way to acquire toxicant is through feeding. 4) We will use large numbers (thousands) of individuals in each "experiment" and unless otherwise stated, each insect is tested individually. 5) Unless stated otherwise, sublethal doses have no effect on the target.

Definitions. 1) Uniform distribution of toxicant is equivalent to all of the following statements: a) 100%

coverage; b) toxicant is maximally dispersed over the treated area; c) distance between molecules of toxicant is the same for all molecules with a variance of zero; and d) toxicant dose in grams per unit area is the same at all spatial scales—hectares to square micrometers. We realize that this is a restrictive definition for uniform. Applying 1 g of toxicant at 1-m intervals in a grid pattern would create a statistically uniform distribution of toxicant if sampling were done over a hectare spatial scale. However, we cannot imagine that this is what is being recommended when a pesticide label suggests to apply the product for "uniform coverage," nor can we imagine that this would be an effective pest control strategy for any product approved for pest control in the United States and used at label rates. Any definition for uniformity requires a description of spatial scale, and we have chosen one definition. 2) Uniform distribution of deposits is not equivalent to uniform distribution of toxicant except when each molecule of toxicant is a deposit. 3) "Very small deposits" are considered smaller than the bite size of the insect larva. As such, the larva always consumes the entire deposit. 4) A "lethal dose" of toxicant is the smallest physical quantity of toxicant required to be lethal. "On balance, it is a reasonable generalization that drops carrying much in excess of a single lethal dose would be wasteful." (Hartley and Graham-Bryce 1980).

Definition of Variables. l is the fraction of leaf consumed. The fraction of leaf remaining is $1 - l$. d is the dose applied in terms of the number of larvae that dose could kill, e.g., $d = 1$ is a dose sufficient to kill one larva and no more. It does not matter if the larva grows, because if the larva survives, then d was <1 , by definition. m is the number of larvae. n is the number of deposits. k is the behavioral response threshold dose below which no behavioral response occurs and above which larvae stop feeding until they recover. As a default, k will equal 1, but otherwise $0 < k < d = 1$.

Model. Redefining the effect of dose on efficacy ($m = 1$): a larva feeding on a leaf uniformly coated with toxicant will eat the entire leaf if the toxicant is only sufficient to kill the larva. Applying toxicant sufficient to kill two larvae will result in one-half the leaf being eaten. If the dose is 4 times that required to kill the larva, then only one-fourth of the leaf will be eaten. So, with uniform toxicant distributions, the fraction of leaf eaten is inversely proportional to the toxicant dose relative to the minimum dose required to kill the larva.

$$l = 1/d \quad [1]$$

However, what if the dose is divided into multiple very small deposits (such that $n = d$)? If $n = 1$, then the larva might eat the deposit with the first bite, or it might eat the entire leaf before acquiring the deposit. Breaking the leaf into bite-size pieces, there will be an equal number of larvae that eat all of the pieces, all but one, all but two, down to a larva that will eat only one. Because both deposit location and feeding location are at random, there should be an equal probability for a larva to eat the deposit after eating all others or to eat

the deposit on the very first try. Likewise for the second to last or the second try, third to last or third, and so forth. As the number of tries approaches infinity, the average quantity of leaf eaten will approach one-half. With $n > 1$ and random placement of deposits on the leaf, the distribution of those deposits might seem aggregated or dispersed. At one extreme, all the n deposits could be deposited one on top of another. In this situation, the deposit would behave exactly as if one had applied only one deposit, and we know that one-half the leaf will be consumed in this case. This is because as a single deposit the probability of acquiring a lethal dose is equal to the fraction of leaf consumed. At the other extreme, deposits could end up dispersed, where each deposit is equidistant from all other deposits. In this case, one could divide the leaf up into equal-sized portions such that each portion contains one deposit. A larva will start feeding in one of these portions, and on average will consume one-half of that portion. So, with sufficient toxicant to kill two larvae distributed as two lethal doses, on average, one-half of one-half of the leaf will be eaten. By extension, if the dose is sufficient to kill m larvae, then

$$l = 1/(2d) \text{ for } n = d \geq m \geq 1 \quad [2]$$

The difference between equations 1 and 2 is shown in Fig. 1. If droplet distribution on the leaf surface is random, we would expect that droplet dispersion would sometimes be aggregated and sometimes dispersed. Under this condition, the average leaf area lost will be half-way between one-half and $1/(2d)$. Assuming $d > 1$, then $1/(2d) < \text{one-half}$, and on average

$$l = \frac{d-1}{4d} + \frac{1}{2d} \quad [3]$$

Therefore, for protecting a crop from damage caused by a single larva, the uniform toxicant distribution will be equal to or better than other distributions if $d \geq 3$. For this experiment, if the leaf cannot withstand 33% defoliation, then one is better off with the uniform toxicant distribution if one has no control over the placement of deposits on the leaf surface. However, these bounds for the heterogeneous toxicant distribution are based on the possibility that all the droplets could land and remain at the same location on the leaf. As the number of droplets increases, the likelihood that all of them will impact the same location becomes increasingly remote, but by similar logic the probability that all droplets will be uniformly dispersed also becomes more remote as the number of deposits increases. Defining the probability that all n deposits will be within a specific distance of one another could be used to provide more realistic bounds to these equations. Furthermore, application equipment has been developed that, in theory, could reduce the clumping of deposits: the electrostatic sprayer. Droplets with the same charge repel each other, thereby reducing the likelihood that they will impact at the same location (Matthews 2000).

What effect does changing toxicant per deposit have on efficacy? Consider a treatment with a dose just sufficient to kill a larva divided into two deposits.

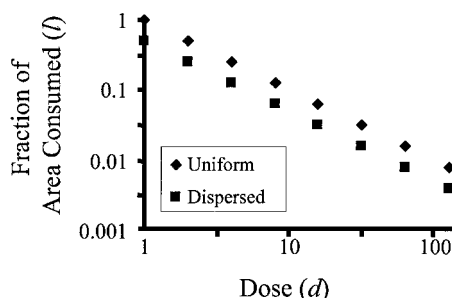


Fig. 1. Difference in leaf area consumed by 1 larva between a leaf protected by a uniform toxicant distribution and uniformly dispersed lethal deposits.

If the deposits were uniformly dispersed, they would each be in different halves of the leaf. The larva would eat one-half the leaf to acquire the first deposit and, on average, one-half of the remaining leaf to acquire the second deposit. So, if it takes n deposits to acquire a lethal dose, then

$$l = 1 - 1/(2n) \text{ for } n > d = m = 1 \quad [4]$$

As n gets large, the toxicant gets more uniformly distributed over the leaf surface, and the quantity of leaf remaining approaches zero. If the deposits were clumped, they would act as a single deposit, with results as discussed. Note: it doesn't make any difference how the lethal dose is distributed between the deposits, because we are still assuming no sublethal effects.

What effect does increasing the number of larvae have? If $d = 1$ and $m = 2$, then the leaf with the uniform deposit will be entirely consumed, and both larvae will survive. If the toxicant is uniformly dispersed and the dose is insufficient to kill all larvae ($d < m$), then all the larvae will live. If the deposit is a single very small deposit, then the leaf will be consumed and one larva will die. With multiple deposits, the effect of multiple larvae depends on their feeding behavior. Such behavior is bounded by two extreme examples: sequential or simultaneous. Sequential feeding is where one larva feeds, and the next starts only after the previous larva dies. This might occur with larvae that are very territorial, where a new larva can move in only if the incumbent dies. Simultaneous feeding is where all larvae feed at the same time and at the same rate. This might occur with gregarious species such as tent caterpillars. If feeding is simultaneous, then on average each larva will consume one-half of its portion of leaf, and therefore one-half the leaf will be eaten, assuming $m = n = d$. If $m < n$, each larva will still eat half of a section of protected leaf where each section is defined by the leaf area divided by the number of deposits, or in general:

$$l = m/(2n) \text{ for } m \leq n \quad [5]$$

For sequential feeding, deposits will protect one-half of an area the size of the remaining leaf divided by the number of remaining deposits. Each larva reduces the number of deposits by 1, and reduces the

Table 1. A table of the area eaten for $n \geq m \geq 4$

	$m = 1$	$m = 2$	$m = 3$	$m = 4$
$n = 1$	$\left(\frac{1}{1}\right)\left(\frac{1}{2}\right) = \frac{1}{2} \Rightarrow \frac{1}{2}$			
$n = 2$	$\left(\frac{1}{2}\right)\left(\frac{1}{2}\right) = \frac{1}{4} \Rightarrow \frac{3}{4}$	$\left(\frac{1}{2}\right)\left(\frac{\left(\frac{3}{4}\right)}{1}\right) + \frac{1}{4} = \frac{5}{8} \Rightarrow \frac{3}{8}$		
$n = 3$	$\left(\frac{1}{3}\right)\left(\frac{1}{2}\right) = \frac{1}{6} \Rightarrow \frac{5}{6}$	$\left(\frac{1}{2}\right)\left(\frac{\left(\frac{5}{6}\right)}{2}\right) + \frac{1}{6} = \frac{9}{24} \Rightarrow \frac{15}{24}$	$\left(\frac{1}{2}\right)\left(\frac{\left(\frac{15}{24}\right)}{1}\right) + \frac{9}{24} = \frac{33}{48} \Rightarrow \frac{15}{48}$	
$n = 4$	$\left(\frac{1}{4}\right)\left(\frac{1}{2}\right) = \frac{1}{8} \Rightarrow \frac{7}{8}$	$\left(\frac{1}{2}\right)\left(\frac{\left(\frac{7}{8}\right)}{3}\right) + \frac{1}{8} = \frac{13}{48} \Rightarrow \frac{35}{48}$	$\left(\frac{1}{2}\right)\left(\frac{\left(\frac{35}{48}\right)}{2}\right) + \frac{13}{48} = \frac{87}{192} \Rightarrow \frac{105}{192}$	$\left(\frac{1}{2}\right)\left(\frac{\left(\frac{105}{192}\right)}{1}\right) + \frac{87}{192} = \frac{279}{385} \Rightarrow \frac{105}{385}$

The area remaining is shown after the arrow; n is the number of deposits, m is the number of larvae.

remaining area by some amount that can be calculated based on equation 2. If $m = d = 3$, then the first larva will eat one-half * one-third of the leaf and leave five-sixths of the leaf protected by two deposits. The second will eat one-half * five-sixths * one-half of the leaf and leave 15/24th of the leaf protected by one deposit. Using this approach, the results for m and n up to four are shown in Table 1. Equation 6 presents a formula for calculating l for any $n \geq m \geq 0$. The equation is not derived directly from previous equations, but previous equations are used to generate a sequence of numbers. Then an equation was generated that exactly predicts any element within this sequence. Table 2 shows how the numerator was generated. The denominator is found in much the same way. Observing that every term in the denominator is created in part by a division by two and each calculation has m terms (expanding Table 1 out fully makes this clear), we factor out 2^m first, to realize that the remaining terms are a factorial with only the first m numbers of the sequence (i and j are integers used as counters).

$$l = 1 - \frac{\prod_{i=1}^m (2n - (2i - 1))}{2^m \prod_{j=1}^i (n - j + 1)} \text{ for } n \geq m > 0$$

[6]

The importance of understanding the feeding behavior of the target population is demonstrated by this difference in outcome for sequential versus simultaneous feeding. Graphically, it is shown for $0 < n < 10$ and $0 < m < n$ in Fig. 2. Populations with solitary larvae would follow the sequential feeding pattern, whereas gregarious insects would be closer to the simultaneous feeding pattern.

Another typical feeding pattern is to eat only part of a leaf, and then move to fresh food. Redefining "leaf" to mean the fraction of the plant potentially consumed

by the larva could cover this condition. An alternate approach would be to redo the previous experiments and remove larvae from the leaf after a specified level of damage (counting live versus dead after consumption of $x\%$ of the leaf area). With uniform toxicant distribution, if there is sufficient toxicant to kill one larva, then mortality will be zero for all $l < 1$. If there is toxicant sufficient to kill two larvae, then all larvae will die if $l = \text{one-half}$. In general, mortality will be 0% if $l < 1/d$ and 100% if $l = 1/d$ for uniformly distributed toxicants. If the toxicant is distributed as a very small deposit, then the probability of encountering the deposit equals the fraction of leaf a larva is allowed to eat, or percentage mortality = l . With multiple deposits, a larva will feed until either it eats a deposit or is removed. Thus, percentage mortality is 100 if $l \geq 1/d$, and $1/d$ for all other cases where $d \geq m = 1$.

Another aspect of how organisms interact with toxicants is in feeding behavior while remaining on a leaf. In crude terms, larvae can be considered nibblers or chompers (Ebert et al. 1998). Nibblers take one mouthful here, move, and feed again. Chompers stay in one place and feed. Consider an experiment where a minimal dose is applied to a leaf surface as a single very small deposit. Because, by definition, the deposit will be eaten in a single mouthful, there will be no difference in the amount of damage caused by nibblers or chompers. However, consider a deposit that takes two bites to consume. If the chomper encounters the

Table 2. Numerators for area remaining from Table 1 on the left side

	$m = 1$	$m = 2$	$m = 3$	$m = 4$	$m = 1$	$m = 2$	$m = 3$	$m = 4$
$n = 1$	1				1			
$n = 2$	3				3	3*1		
$n = 3$	5	15			5	5*3	5*3*1	
$n = 4$	7	35	105	105	7	7*5	7*5*3	7*5*3*1

The sequence at $m = 1$ is $2n - 1$. By breaking the values for $m = 2$ into a product of the numerator for $m = 1$, and breaking down the numerator for $m = 3$ into the product of $m = 1$ and $m = 2$, we see that the numerator is the product of odd integers starting with the value at $m = 1$.

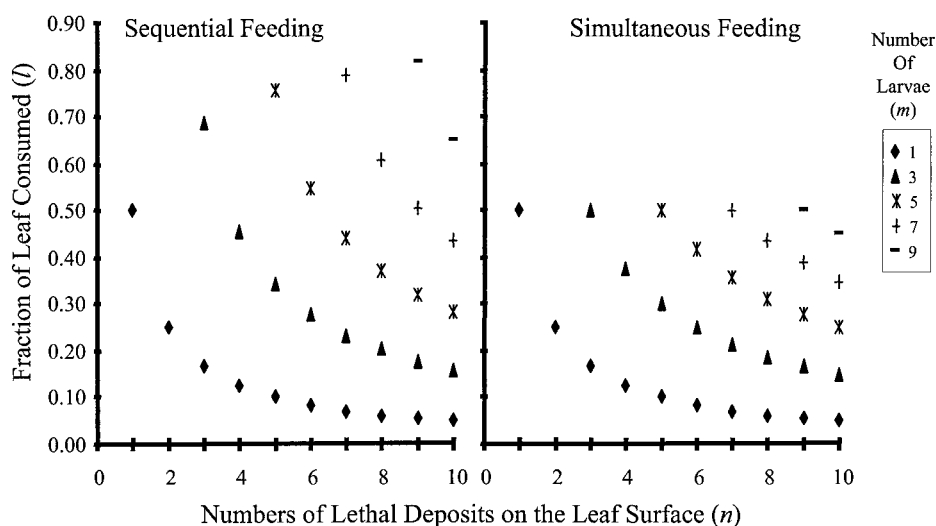


Fig. 2. Difference in damage caused by sequential versus simultaneous feeding behaviors based on the numbers of lethal deposits on the leaf ($n \geq m > 0$).

deposit, it is more likely, on average, to eat the entire deposit. However, the nibbler must encounter the deposit twice to acquire the lethal dose. Consequently, the chomper will eat about one-half the leaf before acquiring the deposit, whereas the nibbler will eat three-fourths of the leaf (one-half the leaf to acquire the first deposit and one-half of the remaining leaf to get the second). However, this assumes random movement. A nibbler may be more (or less) likely than some other nibbler with a different nonrandom behavior pattern to encounter a deposit on which it has already fed. The effect of movement behavior on the probability of encountering adjacent locations can be demonstrated using chess pieces: a bishop finds it impossible to get to a square at the cardinal directions to the one it is currently on no matter how it tries, but a queen at least has a chance given moves of random direction and distance (at worst 1 in 28). In contrast a king has a very good chance (at worst 1 in 8) to get to any specific square adjacent to its current location.

Sublethal effects influence the toxicant-organism interaction. Consider tests that use very small deposits. If a larva has no response up to one-half the lethal dose, and stops feeding thereafter each deposit must contain less than one-half of the total dose (to avoid a sublethal response upon acquisition of one deposit), but more than one-half a dose (to acquire the remaining dose between sublethal and lethal). Because no deposit satisfies both conditions, each dose must contain a lethal dose. In general, the individual dose must exceed $1 - k$ ($k > \text{one-half}$), otherwise the final deposit will be unable to deliver sufficient toxicant to bridge the gap between no response and a lethal response. However, the sum of all doses except the final one must be less than k , otherwise sublethal behavior will be initiated. Furthermore, if $k = 0.9$, then if $d/n = 0.45$ the larva will acquire a sublethal dose (the first bite will have $0.45d$, the second $0.90d$, at which point sub-

lethal behavior will be initiated to eliminate the dose), but if $d/n = 0.6$ or 0.4 a lethal dose will be acquired. These gaps in efficacy are such that any d/n between k/n and $1/n$ will result in a sublethal response.

One could make the sublethal effect more realistic by adding a time component to the model, at least implicitly. The simplest approach is to assume that droplet density is such that the larva will acquire one dose in each time interval that it is feeding. During this time interval, it will also metabolize some quantity of toxicant. If the larva stops feeding until just sufficient toxicant is eliminated to go below the threshold for sublethal behavior, then each dose must contain $1 - k$ toxicant and each dose must contain more toxicant than the larva can metabolize in each time interval.

One additional complication occurs when using any statistical model to analyze data where heterogeneous toxicant distribution plays a role in determining the biological result. The variance structure of such data is not uniform between treatments. For example, the variance in response for the uniform toxicant distribution is zero. For a single insect feeding on a treated leaf containing just sufficient toxicant, the insect must eat the entire leaf to acquire a lethal dose. The entire leaf is eaten, and mortality is 100%. In contrast, larvae feeding on leaves treated with a very small deposit may eat the deposit on the first mouthful or may acquire the deposit only on the very last mouthful. Although mortality has no variance (with a lethal deposit the larva eats until it dies, mortality = 100%, $s^2 = 0$), there is variability in the quantity of leaf eaten ($s^2 > 0$) because the number of bites required to encounter a lethal dose varies with the distribution of deposits and the behavior of the insect. One could rework our equations and start with the assumption that one will allow the larva to feed until it either dies or has consumed some fixed proportion of the leaf. This will result in variability in both mortality and leaf

area consumed. Although we do not develop this model to predict the variance, we have shown the change in variance in biological systems due to changes in heterogeneity of the toxicant distribution (see Fig. 3 in Ebert et al. 1999).

Model Results. 1) For heterogeneous toxicant distributions, a decrease in deposit size will improve efficacy. For homogeneous toxicant distributions, an increase in deposit size will improve efficacy. The boundary is at the point where a single deposit is both small enough to be entirely consumed and large enough to contain a lethal dose. If deposits on plant surfaces are described in terms of their number, size, and toxicant per deposit, then the uniform toxicant distribution can be described with two views: 1) there is a single deposit that covers the entire surface at a low concentration; or 2) there are millions of uniformly dispersed deposits each containing a single molecule of toxicant (maximum concentration). For example, assume that the larva eats the leaf in 20 bites. With view 1, $m = d = 1$ and $l = 1$ at the end of the test. If we shrink the size of the deposit so now it takes 19 bites for the larva to get a lethal dose. The larva eats the first 18, but then there is a 50/50 chance that the last one will have the remaining dose, $l = 19/20 + (\text{one-half})(1/20) = 39/40$. This is true up to the point where the size has decreased to the point where it is a single very small deposit, and we have saved half the leaf. Now, split this deposit into two very small deposits, each with one-half d . Equation 4 states that with one deposit, one-half of the leaf will be lost and as n approaches infinity the leaf area lost will approach 1. As n approaches infinity, the droplet size will get smaller as there will be less and less material in each deposit. So, any experiment examining the effect of deposit size will find that droplets below a certain size will decrease efficacy.

2) Uniform coverage, 100% coverage, or thorough coverage is recommended on many pesticide labels (e.g., Agrimek 0.15 EC, Pyramite, Pounce WSB, Steward, Lannate, Intrepid 80 WSP; Anonymous 2002). If these terms are advising to apply the product such that there is a uniform distribution of toxicant over every surface, then we have shown in the model that this will be wasteful (high dose necessary to protect the leaf) or ineffective (entire leaf is eaten). Alternatively, if these terms mean apply lethal deposits uniformly over the field, then a discussion of spatial scale is needed. One could achieve a uniform distribution by applying one lethal deposit per square meter, and then sampling the field in one square meter sections. This approach would show a uniform distribution, but I hardly think it would be effective. The solution to this problem might be to provide specific application recommendations on the label: apply "our product" to "this crop" at "a dose" in "a volume" with a sprayer producing a "VMD range" and "NMD range" and "droplet velocity range" as is achieved by a standard hydraulic sprayer at "this pressure" with "this spray tip."

3) Deposits that contain less than a lethal dose result in increased time required to acquire a lethal dose, and increased damage to the leaf.

4) Larval behavior both as individuals (as discussed) and collectively (equations 5 and 6 influence efficacy). Larvae that move frequently may, in effect, fragment larger deposits, thereby increasing their chance of survival. Aggregation behavior will alter the acquisition of toxicant for each individual. Damage from aggregated feeding behaviors will not exceed 50% so long as there are at least as many lethal deposits as there are larvae. However, populations exhibiting sequential feeding could cause 100% defoliation, as the number of larvae gets large. Therefore, crop protection should be more difficult for pests feeding sequentially.

5) Behavior of individual insects influences efficacy when larvae must feed on multiple deposits to acquire a lethal dose.

6) Sublethal effects can restrict the effective dose per deposit to a narrow subset of the theoretical maximum range.

7) Uniform toxicant distributions will have the lowest variance in response relative to other toxicant distributions.

8) The relationship between insect mortality and crop protection changes with insect behavior and toxicant distribution.

Discussion

The above-described model is useful because it focuses on the interactions between individual insects and individual deposits. In an agricultural context, this is appropriate because the atomization properties of agricultural spray equipment are described by the number and size of the droplets in the spray cloud; thus, the model explores the interaction in terms relevant to the process whereby we apply toxicants. However, in a field context this model is less useful because it requires information about the insect's environment that is seldom collected. We also recognize that the "real" world has many additional complicating factors: toxicants decay, toxicants act through inhalation and contact, toxicant efficacy is temperature-dependent (Scott 1995), and target health influences toxicant efficacy (Abro and Wright 1989, Mohamad and Van Emden 1989, Verkerk and Wright 1996, Moldenke et al. 1997). Also, these exercises focus on individual outcomes. Populations of organisms may display emergent properties that are not readily apparent from examining the simple underlying rules governing individuals.

In general, the above-described model does not include time as an explicit component of the model. However, we recognize that one of the dominating forces in toxicant-organism interactions is the effect of time. Consider the uniform toxicant distribution: a larva eats one mouthful, and acquires a few molecules of toxin. It takes another mouthful and acquires an equal dose. As it eats the last mouthful of leaf, it acquires a lethal dose and dies. So, mortality is 0% until the very end of the experiment where it jumps to 100%. When graphed, this is an extreme example of the typical dose-response curve. In contrast, the treat-

ment with a single very small deposit has a mortality curve that is just the probability of acquiring the toxicant, and this probability is a function of the feeding rate of the insect and the fraction of the available leaf that has been eaten. When graphed, this is a straight line (Ebert and Hall 1999).

Dose and time are interchangeable quantities for uniform toxicant distributions. Consider the uniform toxicant distribution with a constant feeding rate. Doubling the feeding rate will halve the time it takes to acquire a dose. Doubling the dose will halve the time it takes to acquire a dose. Doubling the time allowed to acquire a dose will permit one to apply only one-half the dose. However, these relationships work only when toxicant is uniformly distributed. If the dose is concentrated as a single, very small deposit, then the only important factor is how long it takes for the larva to encounter and acquire the deposit. Toxicant distributions between these two extremes will benefit to a greater or lesser extent from the exchangeability of dose and time.

Understanding the role of toxicant distribution may be crucial to making better use of new pest management opportunities based on new chemistries or new delivery systems (e.g., transgenic crops). One recent article discussed the qualities of an ideal agricultural spray from a biologist's perspective (Wolf and Downer 1998). Although we have focused exclusively on efficacy, the broader context of spray application will need to be addressed to build a fully optimized system. However, based on this model, we would add to the criteria previously proposed for an ideal agricultural spray: droplets should contain only enough toxin to be lethal, no more, no less. Quantify the maximum tissue loss, double that value and apply one droplet in that area. Alternatively, determine how long it takes the larva to eat some portion of leaf. Given the decay rate of the toxicant, there should be one lethal deposit in every area of leaf that the larva could consume in the time it takes the toxicant to decay.

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